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		Official page numbers:
SPATIO-TEMPORAL CLUSTER OF CASES OF INVASIVE GROUP B *NEISSERIA MENINGITIDIS* INFECTIONS ON THE ISLAND OF MONTREAL	F-1	25 – 28
RESPIRATORY VIRUS SURVEILLANCE	F-4	29 – 30
INSTITUTIONAL OUTBREAKS OF INFLUENZA IN ONTARIO	F-5	30
FOUR OUTBREAKS OF BOTULISM IN UNGAVA BAY, NUNAVIK, QUEBEC	F-5	30 – 32

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SPATIO-TEMPORAL CLUSTER OF CASES OF INVASIVE GROUP B *NEISSERIA MENINGITIDIS* INFECTIONS ON THE ISLAND OF MONTREAL

Over a four-week period between 23 September and 20 October 1995, five cases of invasive *Neisseria meningitidis* infection, including one presumed case and four confirmed cases, were reported to the *Direction de la santé publique de Montréal-Centre* (Table 1). The five cases involved residents of two adjacent areas located north of the centre of the Island of Montreal. Although the cases had not been in direct contact, they were linked socially.

Case 1: The first case involved a 51-year-old female who was living and working as a nurse in a hospital in the area. On 23 September 1995, she felt feverish. The next day she developed petechiae, purpura fulminans, and pains in her legs; she was hospitalized with a diagnosis of pneumonia. Epidemiologic investigation found that the episode had been preceded by an influenza-like syndrome, beginning on 10 September and persisting for 2 weeks. A blood culture revealed the presence of group B *N. meningitidis*. Following treatment with erythromycin and ampicillin, the patient recovered completely. She also received rifampicin prophylaxis, as did her husband and 19-year-old son.

Case 2: The second case involved an 18-year-old female from outside Montreal who was attending an educational institution located in this particular area of the Island of Montreal. She also lived in this area during the week throughout the school year. On 3 October 1995, she developed a fever and headache. The next day, she presented with vomiting, convulsions, and disorientation; her condition deteriorated rapidly. She was taken to the emergency room of one hospital where she showed a lack of response to verbal stimuli and a bilateral Babinski's test, and a reduced response to profound pain. A blood culture was done but, because of her critical condition, the patient was started on six million units of penicillin G and two grams of ceftriaxone; she was immediately transferred to another hospital. A lumbar puncture was performed at the second hospital; the cerebrospinal fluid (CSF) was cloudy and direct examination revealed the presence of 460 leucocytes; all were polynucleated. The patient died within a few hours of admission to the second hospital. The autopsy identified the cause of death as bacterial meningitis. All the blood cultures including the one done on the sample taken prior to initiating antibiotic therapy, CSF cultures, and biopsy cultures were negative. It is believed that she received

Table 1
Cases of group B *N. meningitidis* infections on the Island of Montreal between 23 September and 20 October 1995

Case	Sex	Age	Onset of symptoms (day/month)	Serotype/ Subtype	Electrophoretic complex	Outcome
1	F	51	23/	4:P1.15	ET-5	Recovery
2	F	18	3/10	—	—	Death
3	M	15	7/10	4:P1. negative	ET-5	Recovery
4	F	15	13/10	4:P1. negative	ET-5	Recovery
5	M	19	18/10	4.P1.15	ET-5	Recovery

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meningococcal vaccine during the 1992 immunization campaign. Nearly 20 young people from her residence, including her roommate, her close friends, and individuals who had shared cigarettes, drinks or food with her, received rifampicin prophylaxis.

Case 3: This case involved a 15-year-old male living and attending high school in the same area of the Island of Montreal. On 7 October, he developed fever, pharyngitis, vomiting, petechiae, and disorientation. He was hospitalized the same day with a presumptive diagnosis of *N. meningitidis*, which was confirmed by CSF culture. He recovered completely following medical treatment. Five family contacts, 10 close friends, and six other social contacts received rifampicin prophylaxis.

Case 4: This case involved a 15-year-old female also living and attending high school in that area. In early October, she apparently developed a non-objective fever, a sore throat, and fatigue. On 13 October 1995, she felt feverish again. The next day, she developed a headache, vomiting, a stiff neck, lethargy, and irregular erythematous patches on her back and legs. On 15 October, she visited the emergency room of a hospital and was admitted with a diagnosis of meningitis. She had received Mérieux A-C vaccine during the mass immunization campaign of 1993. Gram-negative diplococci were found in her CSF; culture confirmed the presence of group B *N. meningitidis*. She was treated with ceftriaxone and dexamethasone, and her condition gradually improved. Four family members and seven friends with whom she had shared food and cigarettes received rifampicin chemoprophylaxis.

Case 5: This case involved a 19-year-old male living in the same area of the Island of Montreal. In the fall of 1995, he was working as a security guard. At the end of September, he developed an influenza-like syndrome, which persisted for three weeks. On 18 October, he developed a fever, headache, nausea, vomiting, purpura fulminans, diarrhea, abdominal pains, and pains in the joints and back. On 19 October, petechiae appeared. He consulted a physician and was placed in intensive care in a Montreal hospital with a diagnosis of meningococcemia. His condition was complicated by renal failure and disseminated intravascular coagulation. He also had received Mérieux A-C vaccine during the mass immunization campaign of 1993. He was treated with penicillin, ceftriaxone, and vancomycin. A blood culture revealed the presence of group B *N. meningitidis*. A total of 14 individuals received rifampicin chemoprophylaxis. The patient recovered completely following treatment.

Epidemiologic Links: Cases 3, 4, and 5, as well as the son of Case 1, enjoyed "rave" parties, where young people get together to dance and consume "energizing" or "smart" drinks and sometimes stimulants. Since these are clandestine parties, it was difficult to obtain precise information on specific locations. The son of Case 1 knew Cases 2 and 4, although he had not been in direct contact with them in the preceding weeks. He was a regular patron of the same bar in this particular area of the Island of Montreal as was Case 2. In addition, some of his close friends were also friends with Case 4 and had received rifampicin prophylaxis as a result of having significant contact with her. Cases 4 and 5 lived on the same street, a few houses apart.

Laboratory Results: Four isolates were obtained for detailed characterization and determination of molecular homology. Analyses performed by the *Laboratoire de santé publique du Québec* (LSPQ) and the Laboratory Centre for Disease Control (LCDC) demonstrated that the isolates obtained from four of these cases belonged to the same group and serotype (B:4). Two isolates were subtype P1.15, while another two were P1, negative (Table 1). Because of the spatio-temporal cluster of cases, LCDC was asked to analyze the electrophoretic mobility of cell enzymes[1,2]. The analyses demonstrated that these isolates belonged to the same electrophoretic profile, the ET-5 complex. Finally, pulsed-field gel electrophoresis of the DNA fragments generated by the use of four restriction endonucleases revealed that the four isolates had identical genomic fingerprints[3].

Discussion: The proportion of invasive *N. meningitidis* infections associated with group B to those caused by group C increased in Montreal in 1995 over the period 1990 to 1994. This trend has been evident in the province of Quebec as well since the massive use of meningococcal vaccine (A,C,Y,W-135 or A,C) in 1993 in response to the emergence of a virulent group C clone characterized as ET-15 (F. Gosselin, LSPQ, Ste-Anne-de-Bellevue: personal communication, 1996)[4].

When outbreaks or spatio-temporal clusters of cases occur, identification of the group and serotype is not enough to establish that the same strain is responsible. *N. meningitidis* serotypes can be characterized in greater detail by identifying the subtype, which is determined by the antigenic properties of the class 1 exterior membrane proteins. However, 30% to 50% of the isolates cannot be subtyped, as was the case for two of the isolates obtained in the cluster of cases described in this report. Analyses of the electrophoretic mobility of the cell enzymes permitted determination of the clonal identity of the meningococcal isolates[1]. The laboratory results showed that two phenotype variants of a single clone of *N. meningitidis* of the ET-5 complex were circulating in this area of the Island of Montreal in September and October 1995. In fact, two different subtypes (P1.15 and P1, negative) were identified from the isolates. Gene coding for the class 1 exterior membrane proteins (P1) shows wide variations in expression from one isolate to another[5]. The results of the pulsed-field gel electrophoresis suggest that these isolates are genotypically identical but phenotypically different.

The common factors identified by examination of the cases in this cluster were participation in one or more rave parties and patronage of a specific bar. A recent study suggests that active and passive smokers exposed to *N. meningitidis* have increased susceptibility to the disease; and crowded conditions, which promote droplet transmission, combined with elevated levels of smoking in a bar environment could explain the association of the disease with bar patronage[6]. These are important risk factors and likely played a role in the appearance of the cases in Montreal.

Strains of the ET-5 complex are known for their ability to cause epidemic disease[1]. They were first identified retrospectively in Norway in 1974 and were associated with an epidemic of invasive group B *N. meningitidis* infections, which continued until 1991[7]. These group B meningococcus strains of the ET-5 complex subsequently caused epidemics in a number of other countries[1] and, more recently, in the United States[8,9]. For example, from 1988 to 1990, the Sao Paulo region of Brazil experienced an epidemic of meningococcal disease caused by a clone of the ET-5 complex, which had been present in the region since 1979. The increased incidence of meningococcal disease in this region was associated with the increased prevalence of a single clone (B:4:P1.15) of the ET-5 complex[10].

It is not known how long these strains of the ET-5 complex have been circulating in the Montreal region or in Quebec. This is the first spatio-temporal cluster associated with the ET-5 complex reported in Quebec. The B:4:P1. negative clone has been identified in five other unrelated cases of meningococcal disease in Montreal since January 1995 (F. Gosselin, LSPQ, Ste-Anne-de-Bellevue: personal communication, 1996). However, electrophoretic typing has not been performed on these strains.

It seems clear that epidemiologic investigation of spatio-temporal clusters, and an investigation of the link between the risk factors and the development of invasive infection can permit a better understanding of the evolution of *N. meningitidis* infections. The appearance of cases attributable to the ET-5 complex in Montreal suggests that careful surveillance of group B meningococcal activity and characterization of isolated strains is even more important. Analysis of the electrophoretic mobility of cell enzymes can be a useful tool where spatio-temporal clusters occur.

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Source: *P Le Guerrier, MD, P Pilon, MD, C Sauvageau, MD, D Deshaies, MD, Infectious Diseases Unit, Public Health Branch, Montreal Centre Regional Health and Social Services Board, Montreal, Quebec; A Ryan, National Laboratory for Bacteriology, Bureau of Microbiology, LCDC, Ottawa, Ontario.*

RESPIRATORY VIRUS SURVEILLANCE
FluWatch Project

Figure 1 shows the trend in reported influenza-like illness (ILI) across Canada, as identified through the FluWatch project. Figure 2 shows the standardized rates of ILI across the country by two-week periods. Figure 3 shows the crude rate of reported ILI by age group.

In the United States, the number of states with regional or widespread activity declined in the second week of January. Norway, Netherlands, United Kingdom, and Russian Federation are currently experiencing increased influenza activity. Local outbreaks have also been reported in China, Iran, Israel, and Japan.

This year, the dominant laboratory-confirmed strain has been influenza A/Wuhan/95.

Figure 1
ILI across Canada, reported by FluWatch, 1 October 1996 - 12 January 1997

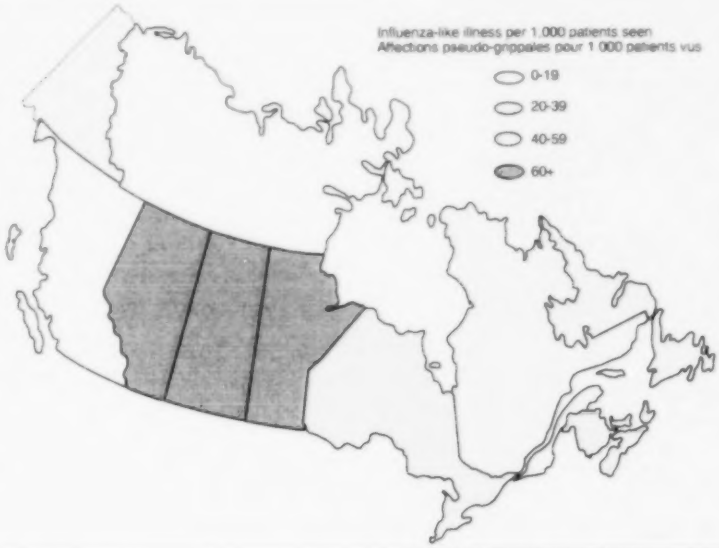


Figure 2
Cumulative rates of ILI across Canada by age group, reported by FluWatch, 1 October 1996 - 12 January 1997

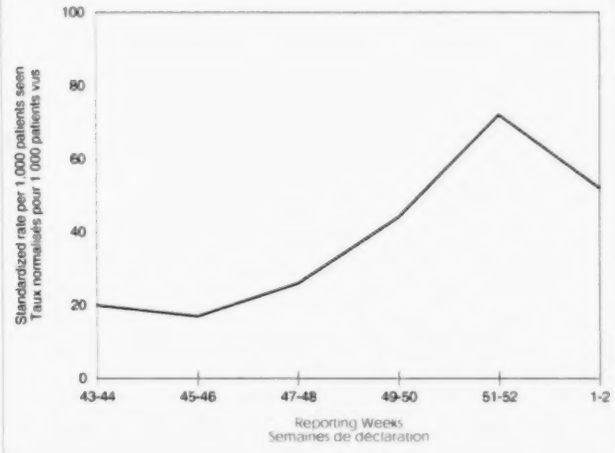
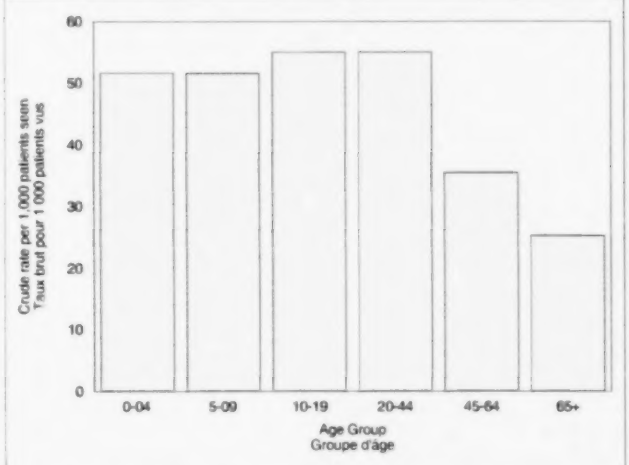


Figure 3
Standardized rates of ILI across Canada by two-week periods, reported by FluWatch, 19 October 1996 - 19 January 1997 (standardized to Canadian population)



Source: *Division of Disease Surveillance, Bureau of Infectious Diseases, LCDC, Ottawa, Ontario.*

INSTITUTIONAL OUTBREAKS OF INFLUENZA IN ONTARIO

The Public Health Branch of the Ontario Ministry of Health, with the help of local health units across the province, conducts an influenza surveillance program every year from November to April. This preliminary report summarizes institutional outbreaks of influenza from early December 1996 to mid-January 1997 in Ontario. This year, influenza activity increased significantly during December 1996 and early January 1997.

Influenza-like illness is defined by the presence of fever > 38° C, cough or sore throat, muscular pain, malaise, and fatigue. From 23 December 1996 to 14 January 1997, 64 institutional outbreaks of influenza (compared to 18 reported during the entire 1995-1996 season) in facilities housing 7,280 residents were reported from 24 of the 42 provincial health departments. The onset date ranged from 7 December 1996 to 6 January 1997. Cough, fever, malaise, runny nose, sore throat, and fatique were the commonly reported symptoms. Thirty-six outbreaks (56%) occurred in nursing homes, 18 in homes for the aged, and seven in retirement homes. The mean attack rate in the 64 outbreaks was 22% (median 19.5, range 2.2% to 71%). Laboratory testing by rapid antigen detection or culture of specimens has confirmed that these outbreaks were due to influenza A. There were 26 deaths among residents for a case-fatality rate of 1.9%. Use of amantadine was reported in at least 17 outbreaks. Among the affected facilities where influenza vaccination rates are known, the rates are generally more than 80%, with some reporting 100%.

Source: *S Neamatullah, MD, Field Epidemiology Training Program, LCDC, Ottawa; M Naus, MD, Disease Control Service, Ontario Ministry of Health, North York, Ontario.*

FOUR OUTBREAKS OF BOTULISM IN UNGAVA BAY, NUNAVIK, QUEBEC

Four unrelated outbreaks of botulism were confirmed in Ungava Bay, Nunavik, in the summer and early fall of 1995.

The first outbreak occurred in Kangiqsualujjuaq, a community of 600 inhabitants in the Ungava Bay area of Nunavik. On 11 July, a 65-year-old male was brought to the local nursing station approximately 16 hours after having shared a meal, with family members, of fermented seal meat, dried seal meat, and aged seal oil. The first symptom was profound weakness followed by a brief syncopal episode. Other symptoms included blurred vision and occasional diplopia, dryness of the mouth, dysphonia, and dysphagia. Unreactive mydriasis as well as paralytic ileus were noted. The patient was sent to the regional hospital in Kuujjuaq where he received three vials (5,000 I.U. each) of type E botulinum antitoxin. He was transferred to Montreal the following day. A total of 24 days of hospitalization was required, due in part to unrelated conditions. Gastric fluid, fecal, seal oil, and seal meat specimens were negative for botulinum neurotoxin. However, the gastric fluid, fecal, and seal oil samples contained viable *Clostridium botulinum* type E organisms. No other participant at the meal developed symptoms.

The second outbreak occurred in Kuujjuaq, a community of 1,500 inhabitants. On 9 August, two Inuit, aged 52 and 23, presented at Ungava Hospital, complaining of abdominal pain, vomiting, nausea, diarrhea, dryness of the mouth, dysphonia, dizziness, and blurred vision. They reported that on the previous day they had consumed approximately 250 g of fermented seal meat. The meat had been left in a closed glass container at ambient temperature for 3 to 4 days before consumption. Monovalent type E antitoxin (three doses of 5,000 I.U.) was administered to the two patients; 12 to 36 hours later both were clinically improving. Some degree of abdominal ileus was observed during hospitalization. On 15 August, the sixth day of hospitalization, they were discharged with residual fatigue and slight dysphagia. The lesser affected patient had been part of a documented episode of botulism in 1975.

Botulinum toxin type E and viable *C. botulinum* were confirmed in the seal meat. Viable *C. botulinum* and botulinum neurotoxin were recovered from a fecal sample taken from one patient 24 hours after the implicated food had been consumed. Botulinum neurotoxin was detected in the serum of the second patient and viable *C. botulinum* was isolated from his gastric contents. Two other people had eaten some of the seal meat. A 6-year-old who had eaten a very small amount remained asymptomatic. The other individual, aged 48, experienced growing abdominal discomfort, dryness of the mouth, and sore throat. One day after the admission of the first two patients, this patient received antitoxin and was hospitalized for 2 days. Serum samples from this patient were negative for botulinum neurotoxin. No other clinical samples were available.

The third outbreak occurred in Tasiujaq, a small community of 140 inhabitants, located 100 km northwest of Kuujjuaq. On 14 August, five individuals ranging in age from 33 to 71 years of age, were transferred from the local nursing station to the Ungava Hospital in Kuujjuaq. Initial complaints included nausea and vomiting for all five patients. Three of the patients developed blurred vision, diplopia, dryness of the mouth, dizziness, and unreactive mydriasis. These were followed by mild hypotension and some degree of respiratory difficulty. All five patients had consumed a meal of fermented walrus meat. A total of 23 participants had consumed various amounts of the meat. The walrus was killed at the end of July and approximately 10 kg of skin, blubber, and meat were kept in a closed plastic container in a warehouse at ambient temperature until consumption on 13 August. Two of the five patients remained in hospital in Kuujjuaq where they received trivalent antitoxin. They were discharged on the fourth day after they had been admitted. The remaining three

patients received two doses of type E botulinum antitoxin and were transferred to Montreal for intensive care. After 48 hours, two of these patients were released from the intensive care unit. They experienced residual asthenia, dysphagia, and mydriasis for 10 days after admission to hospital. The remaining patient remained in intensive care for 10 days. This patient required mechanical ventilation for 9 days, vasopressive medication for 6 days, and parenteral alimentation for 10 days. Digestive functions were particularly slow to recover. Type E botulinum neurotoxin was detected in, and *C. botulinum* type E was isolated from, the walrus meat. Viable *C. botulinum* type E was detected in gastric fluids and fecal samples from three of four patients (a fecal sample was not obtained from one patient).

The fourth, and last, outbreak for 1995 occurred in Kuujjuaq on 1 October, when a 59-year-old female was hospitalized with upper abdominal discomfort, progressive asthenia, postural dizziness, and shortness of breath. On 28 September, the patient had eaten from 100 g to 200 g of fermented seal meat. This food was part of a meal shared with three other people who had consumed smaller amounts and remained free of symptoms. The patient received a total of five doses (5,000 I.U. each) of botulinum antitoxin type E and was hospitalized for 3 days. Residual asthenia persisted for almost 3 weeks. Serum and fecal samples were negative for botulinum neurotoxin and for *C. botulinum*. However, the seal meat was positive for type E botulinum neurotoxin and *C. botulinum* type E.

Source: *J-F Proulx, MD, Coordinator of Infectious Diseases, and V Milor-Roy, Department of Public Health, Nunavik Regional Board of Health and Social Services, Kuujjuaq, Quebec; J Austin, PhD, Botulism Reference Service for Canada, Bureau of Microbial Hazards, Food Directorate, Health Protection Branch, Health Canada, Ottawa, Ontario.*

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